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THE VETERINARY JOURNAL

A MONTHLY REVIEW OF VETERINARY SCIENCE

Est. 1875

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The Veterinary Journal.

SEPTEMBER, 1936.

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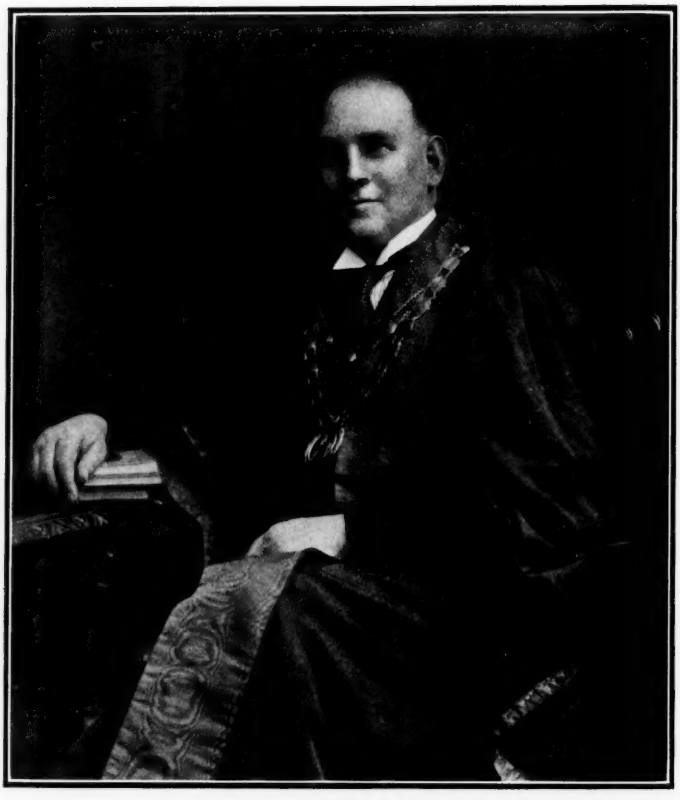

Mr. J. R. Barker, M.R.C.V.S., D.V.H.

President of the National Veterinary Medical Association.

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Veterinary Adviser to various Overseas Governments; late Chief Veterinary Officer to the Corporation of the City of Londo

SEPTEMBER, 1936

Editorial

THE ENGLISH-SPEAKING VETERINARY CONGRESSES.

THE following is the programme of the National Veterinary Medical Association Congress to be held at Scarborough from September 14th to 18th, 1936:

Sunday, September 13th.—8 p.m., Informal "at home" (Royal Hotel).

Monday, September 14th.—10 a.m., Civic welcome by the Mayor of Scarborough (Public Library). 10.30 a.m., Opening of Exhibition of Surgical Instruments, Drugs, etc., by the President (Harrison Room, Public Library). 11 a.m., Paper: "Hysteria in Dogs," by Mr. H. R. Hewetson, M.R.C.V.S. Discussion to be opened by Major F. Chambers, O.B.E., F.R.C.V.S. (Public Library). 8.30–12 p.m., Civic reception and dance (Royal Hotel).

Tuesday, September 15th.—10 a.m., Paper: "Enzootic Abortion in Ewes," by Professor J. Russell Greig, Ph.D., M.R.C.V.S. Discussion to be opened by Capt. T. J. Bosworth, B.Sc., M.R.C.V.S., D.V.S.M. (Public Library). 11 a.m., Paper: "A Consideration of the Accredited Producers' Scheme of the Milk Marketing Board," by Major D. S. Rabagliati, O.B.E., B.Sc., F.R.C.V.S., D.V.S.M. Discussion to be opened by Mr. H. W. Steele Bodger, M.R.C.V.S. (Public Library). 3 p.m., Operations and Demonstrations (Corporation Stables). 8.30 p.m., President's Reception (Royal Hotel).

Wednesday, September 16th.—10 a.m., Paper: "The Detection of the Tuberculous Cow," by Capt. H. L. Torrance, M.R.C.V.S., D.V.S.M. Discussion to be opened by

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Mr. J. S. Steward, F.R.C.V.S., D.V.S.M. (Public Library). 11.30 a.m., Paper: "Some Observations on the Use of Vaccines in the Protection of Cattle against Tuberculosis," by Mr. R. E. Glover, B.Sc., M.R.C.V.S. Discussion to be opened by Mr. H. G. Lamont, M.Sc., M.R.C.V.S. (Public Library). 11.30 a.m., Meeting of Ladies' Veterinary Benevolent Guild at the Royal Hotel. 12.45 p.m., Congress photograph. 2.30 p.m., Council Meeting (Public Library). 3.30 p.m.—Annual General Meeting (Public Library). 7 for 7.30 p.m., Annual Banquet (Royal Hotel).

Thursday, September 17th,—10.30 a.m., Address and Demonstration on "Sterility in Bulls," by Professor Nils Lagerlöf, of Stockholm (Public Library). 2 p.m., excursion to Ravenscar *via* Olivers Mount, Hackness, Harwood Dale, etc., with tea at Raven Hall Hotel, Ravenscar. 8 p.m., Dance in aid of the Ladies' Benevolent Guild (Royal Hotel).

Friday, September 18th.—10 a.m., Paper: "A General Consideration of the Disease Conditions mentioned in the Horse Breeding Acts," by Professor W. M. Mitchell, M.C., M.B., Ch.B., B.Sc., M.R.C.V.S. Discussion to be opened by Major Brennan DeVine, M.C., F.R.C.V.S., D.V.S.M., F.R. San. I., and Major A. A. Pryer, D.S.O., M.R.C.V.S. (Public Library). 3 p.m., Closing Meeting (Public Library).

As in other years, we welcome a distinguished foreigner, and on this occasion we can assure our Swedish guest, Professor Nils Lagerlöf, of the Stockholm Veterinary School, a hearty welcome. His lecture and demonstration of the methods adopted in that country against sterility of bulls will be attended by many country practitioners, to whom it will appeal and be of practical help.

Last month our *confrères* in America held their Seventy-third Annual Veterinary Congress at Columbus, Ohio, and it is interesting to see and compare the programme placed before its members. As with ourselves—but on a somewhat larger scale—each branch of veterinary medicine and surgery is catered for; and there is a well-run social side to which ladies are specially invited—the benevolent and charitable aspect on behalf of those who have fallen upon evil times not being forgotten.

The details of each respective section were as follows:

General Session.—The American Stockman and the Veterinary Profession; Tularemia (illustrated); Progress Report on Crystal-Violet Vaccine Against Hog Cholera; Studies on Gastric Digestion in Ruminants (illustrated).

Section on General Practice.—Control of Parasites in Sheep; The General Practitioner; Diseases of Sheep; Swine Diseases; Arthritis of the Horse; Mastitis in Cattle; Anaplasmosis of Cattle; Pyelonephritis in Cattle and Horses.

Section on Sanitary Science and Food Hygiene.—Live Stock Loss Prevention; Regulating the Sale and Use of Biological Products Containing Pathogenic Organisms; Food and Drug Administration, U.S. Department of Agriculture, Washington, D.C.; Auction and Community Sales; Infections Transmitted in Milk; Meat Inspection. Report of Special Committee on Meat Hygiene: (*a*) Municipal Meat Inspection; (*b*) State Meat Inspection; (*c*) Poultry Inspection; (*d*) What Our Veterinary Colleges are Doing to Prepare Veterinarians for Public Health Work; (*e*) Recommendations of the Special Committee on Meat Hygiene; (*f*) What Canada is Doing Along Meat Inspection Lines.

Section on Research.—Studies on Bovine Blood—The Sedimentation Rate and Percentage Volume of Erythrocytes in Normal Blood; The Pathogenesis of Ketosis—

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Pregnancy Disease of Sheep ; The Significance of Suspicious Agglutination Reactions for Bang's Disease ; The Significance of Pasteurella Boviseptica Encountered in the Blood of some Florida Cattle ; The Problem of the Vectors of Anaplasmosis in the United States ; Infectious Diseases of Domestic Rabbits.

Section on Small Animals.—A Consideration of Heartworm and Tapeworm Infestations in the Dog ; The Prostate Gland of the Dog ; The Diagnosis, Prognosis and Treatment for Hip Injury ; A Study of Streptococcic Infections in Dogs ; Some of the Pathological Lesions Associated with Indigestion in Dogs ; Observations on Canine Piroplasmosis ; The Utilisation of Sheep in the Rabies Vaccine Protection Tests.

Section on Poultry.—Infectious Enterohepatitis ; Salmonella Infections in Poultry ; Studies on the Egg Propagation of Laryngotracheitis and Fowl-Pox ; Relative Values of Farm Grains in Poultry Nutrition ; The Results of the Practical Application of Pullorum Disease Control Methods ; Fowl Leukosis ; Field Observations on the Incidence of Fowl Leukosis in Hatching Eggs and Chicks ; The Cultivation of the Virus of Chick Bronchitis ; The Importance of Endotoxin in the Moderation of Fowl Paralysis by Bacteria of the Paratyphoid Group.

Clinic.—The clinic was held in a well-adapted building within ten minutes' drive of the Convention headquarters. The following outline gives some idea of the scope of the clinical cases that were presented : *Horses.*—Five subjects were scheduled for presentation by eight demonstrators. *Cattle.*—Eight subjects were scheduled for presentation by thirteen demonstrators. *Sheep.*—Seven subjects were scheduled for presentation by ten demonstrators. *Swine.*—Eleven subjects were scheduled for presentation by eleven demonstrators. *Poultry.*—Eleven subjects were scheduled for presentation by twelve demonstrators. *Small Animals.*—Ten subjects were scheduled for presentation by nine demonstrators. In addition there were lectures and demonstrations several times a day on the following subject headings : (1) Mastitis. (2) Chemical determinations as applied to blood, urine and milk, having diagnostic and clinical significance. (3) Demonstration of agglutination test for Bang's disease. (4) Laboratory determinations employed in small animal practice. (5) Laboratory section on general pathology. (6) Laboratory section on parasitology. Parasitic exhibit.

As with the Congress of the National Veterinary Association, the various drug and instrument firms illustrated the most up-to-date advances in scientific equipment in connection with veterinary medicine and surgery, and such things as the latest anæsthetic and pharmaceutical preparations, sera, vaccines, instruments, etc.

The educational value of these Congresses cannot be overestimated—especially for the older and single-handed practitioner who cannot spare the time—because he is busy—to come back to College for a few weeks and go through a post-graduate refresher course. Even if only for a week or two at a time, there is no question as to the value of brief College courses of this kind to the whole-time official on home leave, and especially to the Army or Colonial officer whose retiring age is now at hand; as, with perhaps a family to educate, he knows full well that his pension, valuable as it may appear to be when he is entering the service, will barely keep body and soul together unless it can be supplemented by some outside source on return to civil life.

It is worth while here, too, to draw the attention of those on this side of the

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Atlantic to the use which our American colleagues have put certain surplus funds left over from the Twelfth International Veterinary Congress, which have been invested for the purpose of establishing the following prizes: (1) A $5,000 endowment to a member of the American Veterinary Medical Association for the most noteworthy contribution to the advancement of veterinary science during the preceding year. (2) A $3,000 endowment for the most noteworthy contribution by a veterinarian since the last Congress. Although these are only allowed to be competed for by members of the American Veterinary Association, it is an example which should be followed if a similar opportunity can be made with our own Society.

THE TRADE EXHIBITION AT THE CONGRESS.

NOT the least interesting part of this gathering is the exhibition of the specialities and commodities put up by the many eminent commercial houses catering for the profession in this country and abroad. We feel sure that our readers when visiting the Exhibitors' Hall will find much of interest to them, for the products of these firms have reached a high measure of excellence. We make every endeavour to ensure that only the advertisements of reliable houses are inserted in THE VETERINARY JOURNAL, and in many cases the co-operation of these firms with the profession has been of invaluable assistance. Amongst those exhibiting will be the following well-known concerns, to whom a visit, we feel sure, will be amply repaid: The Anglo-American Pharmaceutical Co., Ltd.; Messrs. Armour & Co., Ltd.; Messrs Arnold & Sons; Messrs. Baillière, Tindall & Cox; British Glues & Chemicals, Ltd.; Messrs. Genatosan, Ltd.; Messrs. C. J. Hewlett & Son, Ltd.; Messrs. Hough, Hoseason & Co.; The International Serum Co., Ltd.; Messrs. Parke, Davis & Co.; Sandoz Products; Willington Medical Products, Ltd.; Willows, Francis, Butler & Thompson, Ltd.; Chas. Zimmermann & Co., Ltd.

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General Articles

THE PRACTICE OF VETERINARY SCIENCE IN GERMANY.

THE number of German veterinary surgeons is about 6,000, who, partly as practitioners in private practice, partly as officials in abattoirs, in official institutes, or as veterinary officers, put their science at their country's disposal. As a result of the increase of the Army, a greater demand for Army veterinary surgeons arose, so that many young veterinary surgeons were engaged for the Army. For this purpose a military veterinary college was opened in Hanover, connected with the ordinary veterinary college, for the education of military veterinary surgeons only. At the same time nearly 2 per cent. of these veterinary surgeons were obliged to withdraw in consequence of the new German law of citizenship, or were limited to private and curative practice only. The economic position of German veterinary surgeons can be called a good one, for besides private practice there are many public institutions which guarantee the practitioner a certain means of existence. First in importance comes the meat inspection and trichina examination, which is solely in the hands of veterinary surgeons in towns. According to more recent regulations, meat inspection must be in the hands of qualified veterinary surgeons in all towns of over 4,000 inhabitants and in health resorts, just as the direction of abattoirs should be in the hands of veterinary surgeons. The next important sphere in which veterinary surgeons are engaged is the eradication of tuberculosis, which is now carried out thoroughly on all the German cattle stocks.

The work of veterinary surgeons consists of clinical examination of the animals standing under control and the report of suspicious animals to the veterinary officer ; the latter issues further measures and puts this stock in quarantine. The control takes place as a rule twice a year. The next important point is the control of milk, and connected therewith the control of dairies which produce special milk, such as certified or graded. The veterinary surgeon has to examine the cows, especially the milk organs, and is responsible for regular samples of milk from every animal. In recent years in this connection there has arisen the Bang method of combating the abortion infection. Here every animal must be examined and samples of blood tested at the so-called " Tierseuchenamt " (animal disease office). Further, any animal exposed for sale, or intended for sale, in course of transport for markets or auctions, must be examined by a veterinary surgeon and its health attested. That vaccination, especially with living cultures, lies in the hands of veterinary surgeons is a matter of course, so that vaccination against red murrain, pig disease or plague, foot-and-mouth disease, or *abortus* (Bang), is exclusively a domain for veterinary surgeons. Through the new law being enforced for the prevention of cruelty to animals,

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by which every painful operation must be made under anæsthetics, the veterinary surgeon has another opportunity to extend his practice. As narcotics and anæsthetics are, as a rule, only available for medical men, many operations (castrations) which used to be performed by unqualified men must now be given to veterinary surgeons. Moreover, in certain parts of Germany methods of combating the breeding of disease and sterility have been developed with State aid and the aid of " Reichsnahrstand " Board of Agriculture. Thus qualified veterinary surgeons are offered a large sphere of work here, and these methods are likely to spread over the whole country.

In conclusion, when summing up the methods which have arisen, with Government aid, from the point of view of the veterinary surgeon, one can confidently say that German veterinary surgeons, besides their ordinary practice, have obtained a wide field of activity in official posts; and according to their qualifications can find an assured means of existence.

OBSERVATIONS ON FOWL PARALYSIS (LYMPHOMATOSIS).

By Major T. DALLING, M.R.C.V.S., and Miss G. H. WARRACK, B.Sc.

Wellcome Physiological Research Laboratories, Langley Court, Beckenham, Kent.

ALL the histological examinations were carried out by Dr. C. L. Oakley.

In connection with fowl paralysis (lymphomatosis), during the past two and a-half years we have carried out investigations into (*a*) artificial transmission; (*b*) infection by contact; (*c*) the occurrence of lymphomatosis in the progeny of selected birds. At autopsy, various tissues, including nervous structures, were examined, and a diagnosis of lymphomatosis is based on the finding of accumulations of lymphoblasts, including plasma cells more extensive than usual. The limits of normality are naturally a matter of personal opinion, but a positive diagnosis was not made unless the accumulations were of such a nature as to produce changes in the surrounding tissue. We have recorded lymphomatosis in any organ or tissue including the nervous system, for we believe that " fowl paralysis " is but a phase of general lymphomatosis.

In the past most observers have terminated their experiments after a few months, or, as we did in experiments (*a*) and (*b*), after a year. From our results in experiment (*c*) it is evident that experiments in connection with lymphomatosis cannot be said to be completed until the birds concerned have actually died. In considering the results of (*a*) and (*b*), the above points must be kept in mind.

Artificial Transmission.

Several workers have recorded the results of their attempts to set up lymphomatosis by the use of tissue of affected birds. In 1933, at the Fifth World's Poultry Congress, Rome, we pointed out that in untreated control birds from the same

group as those treated, we invariably found cases of lymphomatosis, though in smaller numbers than in the treated birds. In the work to be recorded now, we used birds from two different sources. One lot called " susceptible " was bred from stock among which lymphomatosis was present. The other, bred from stock among which we were informed no lymphomatosis was or had been present, we called " resistant."

We used 193 " susceptible " and 187 " resistant birds. They were obtained as day-old chicks, and were reared under hovers in units of eighty, were kept in brooder houses 10 in. × 8 in., and were allowed out on grass runs when ten days old. The two groups were kept on different parts of the farm and had different attendants.

When about eight weeks old the chicks were divided into small groups (equal numbers of cockerels and pullets). The material for injection was (*a*) brain, affected peripheral nerves and spleen of nine affected birds from five different sources showing early and late symptoms, and any tumorous lymphomatic cells found in the affected birds were also used in the mixture ; (*b*) brain, nerve and spleen from normal healthy birds. All tissues were examined histologically, and the presence or absence of lymphomatosis confirmed. The tissues were ground up in fine sand, emulsified in saline, filtered through cotton wool and injected in a dose of a c.c. intraperitoneally. The small experimental groups were kept separately and had different attendants. The injected birds were kept under observation for twelve months, when all were killed and examined. Birds dying in the interval were also examined.

Results.

(1) " Susceptible " stock (Rhode Island Red × Light Sussex) : (*a*) Injected with " fowl paralysis " tissue ; 55 chicks treated ; lymphomatosis found in 14 (22 per cent.). (*b*) Injected with " normal " tissue : 59 chicks treated ; lymphomatosis found in 6 (10 per cent.). (*c*) Control, untreated birds : 53 chicks kept as controls (17 were drowned in a flood) ; no evidence of lymphomatosis found in any bird.

(2) " Resistant " stock (three breeds : Anconas, Buff Rocks, and White Leghorns). (*a*) Injected with " fowl paralysis " tissue : 57 chicks treated (Anconas, 19 ; Buff Rocks, 21 ; Leghorns, 17) ; lymphomatosis found in 4 (Anconas, 0 ; Buff Rocks, 2 ; Leghorns, 2) (7 per cent.). (*b*) Injected with " normal tissue " ; 53 chicks treated (Anconas, 19 ; Buff Rocks, 19 ; Leghorns, 15) ; lymphomatosis found in 3 (Anconas, 1 ; Buff Rocks, 0 ; Leghorns, 2) (6 per cent.). (*c*) Control, untreated birds : 56 chicks (Anconas, 23 ; Buff Rocks, 16 ; Leghorns, 17) ; lymphomatosis found in 4 (Anconas, 2 ; Buff Rocks, 0 ; Leghorns, 2) (7 per cent.).

It is difficult to know what significance to attach to these results. Experiment (1) appears to show that the controls were free from " infection," that the injection of normal tissue caused the disease in 10 per cent., and that the injection

of infected tissue caused 22 per cent. of "infection." These results appear significant, yet in Experiment (2) the three groups showed an equal percentage. It would appear, therefore, that the strain of birds used in the experiments was of importance.

In group 2, the 166 chicks included three breeds; an analysis showed:

Injected	Per cent. affected		
	Anconas	Buff Rocks	White Leghorns
"Fowl paralysis" tissue	0	9.5	11.8
Normal Tissue	5.2	0	13.3
Control, uninjected	8.7	0	11.8
Total, irrespective of injection	5.0	3.5	12.0

The White Leghorn stock appeared to be more susceptible to the disease than the other two breeds. We may mention that all the birds were reared under similar conditions. Three different attendants were in charge of the three groups.

Infection by Contact.

Some workers have concluded that fowl paralysis can be spread by contact from infected to healthy birds, and that it is only to chicks during the first few weeks after hatching that infection can be passed, though the infecting birds may be of any age. We carried out two sets of experiments:

Experiment A.

During the incubation of chicks from eggs laid by susceptible and resistant pullets, four lots of eggs—two from susceptible and two from resistant birds—were in contact in the incubator, and the chicks hatching therefrom were kept and reared together. Other batches of eggs from the two groups were incubated separately, and the chicks produced were reared separately. When about fourteen weeks old the chicks were mixed. The remaining birds are now 18 to 20 months old, and the following are the results:

Mixed Groups.—One hundred and eleven resistant chicks yielded eight cases of lymphomatosis (7.2 per cent.). The 148 susceptible chicks in contact with them yielded 32 cases (22 per cent.).

Unmixed Groups.—The disease occurred in three (4.5 per cent. of 66 resistant chicks), while nine cases (19 per cent. occurred amongst 48 susceptible chicks).

Experiment B.

For this experiment folding units were used and observations were carried out for twelve months.

Susceptible Stock.—Twenty-six chicks were kept in contact with several typical cases of lymphomatosis; three birds (11 per cent.) became affected.

Resistant Stock.—Twenty-one chicks were treated as above, three birds (14 per cent.) became affected.

In neither experiment are the differences wide enough to indicate that infection occurs by contact.

The Occurrence of Lymphomatosis in the Progeny of Selected Birds.

Various workers have suggested that either the condition lymphomatosis is transmitted, or there exists a hereditary difference in susceptibility or resistance. We bred groups of birds to test this hypothesis. The following general observations may be of interest :

(1) Both cockerels (34 cases) and pullets (94 cases) became affected.

(2) Lesions were found in the nervous tissues only of 49 birds ; tumour formation in tissues other than those of the nervous system in 59 ; in both the nervous and other systems in the remaining 20 birds.

(3) Lesions were found in the following tissues ; Brain, 5 ; lumbo-sacral plexus, 31 ; brachial plexus, 35 ; sciatic nerve, 10 ; crural nerves, 3 ; nerves of the sympathetic system, 10 ; brachial nerve, 13 ; costal nerve, 1 (in 16 cases the brachial plexus alone was affected ; in 12 cases lumbo-sacral plexus only ; while in 14 birds both brachial and lumbo-sacral plexuses were involved ; in 30 cases nerve lesions were unilateral) ; liver 36 ; ovary, 24 ; spleen, 9 ; kidney, 16 ; lungs, 5 ; skin, 5 ; mesentery, 9 ; testes, 4 ; pancreas, 3 ; heart muscle, 9 ; pectoral muscle, 1 ; gizzard wall, 4 ; wall of intestine, 1 ; proventriculus, 1 ; oviduct, 1 ; (in 26 birds, one organ only was affected). In those birds no record of " eye " changes are given.

(4) It has often been stated that intestinal parasitic infestation, especially coccidiosis, plays a considerable part in the causation of lymphomatosis. We are inclined to disagree with this view. It happened that in the course of some investigations on coccidiosis, a group of about 30 birds was kept for nine months under conditions which prevented entirely any parasitic infestation, proved by the examination of excreta at short intervals and the killing of some birds periodically for examination. During the period of observation, four out of the 30 birds developed typical lymphomatosis (including nerve lesions), the condition being confirmed histologically. (This observation was made by our colleague, Miss A. W. Greener, M.R.C.V.S.)

In 105 of the 128 autopsies no intestinal parasites or lesions which may have been caused by their presence at any time were found. Coccidia were found in 5, round worms in 9, tapeworms in 8, and both round worms and tapeworms in 1. In none was there any evidence of lesions produced by their action. The incidence of parasites was greater in the autumn months.

(5) The ages of affected birds, together with the types of lesion found, were as follows :

Age.	No. of birds.	Types of lesions. Nerve.	Tumour.	Nerve and Tumour
2–6 months	7	2	3	2
6–12 ,, ...	55	23	20	12
12–18 ,, ...	46	18	23	5
18–24 ,, ...	17	6	10	1
24–28 ,, ...	2	—	2	—
38 ,, ...	1	—	1	—

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We have found that symptoms and lesions may appear in cockerels only after mating, and in some cases after the mating season is over.

Breeding Results.

Four groups of pullets were used, and for mating in 1934 four selected cockerels.

PULLETS.

Group a.—White Leghorns bought as day-old in 1933 and reared by us. This group came from birds which, as far as our knowledge went, had no lymphomatosis in the progeny or family and which had no contact with affected birds.

Group b.—White Wyandottes procured as mature pullets. They were selected not because of any definite clinical evidence of fowl paralysis, but because the iris was unusually light-coloured or the pupils irregular, some being pinpoints. Much culling had taken place in the original flock, and we have diagnosed lymphomatosis in Rhode Island–Wyandotte crosses on the farm.

Group c were Rhode Island Red mature pullets. Some of their sisters had been affected with lymphomatosis. It was believed that the condition had been transmitted to these birds through an imported sire.

Group d consisted of two pullets (sisters) which had been bred from a hen 50 per cent. of whose progeny had shown lymphomatosis during one season. They were R.I.R.'s, and were bred by us.

COCKERELS.

The cockerels used were of two types: (*a*) Nos. 711 and 712, so-called "susceptible.' No. 711 was the sire of the pullets in Group *c* and the sire of No. 712. (*b*) Nos. 203 and 364. These were so-called "resistant" birds, being members of families in which fowl paralysis was apparently absent. They were brothers of the pullets in Group *a*.

Table I shows the group results of the various matings. In Table II are the results of the matings of the pullets in the various groups, irrespective of the cockerels used, and Table III shows the results of matings of the cockerels irrespective of the pullets used.

An interesting point shown in Table IV is the striking difference in the incidence of lymphomatosis in the progeny of individual pullets, for though the groups were chosen because of certain common features, yet the individual members did not all behave alike. Though the records show difference in the incidence of lymphomatosis in the progeny of the different cockerels used, both when the groups and individuals are considered, it is very doubtful whether the difference is significant. (It must be noticed, however, that the selection of cockerels was a matter of some difficulty, and though we assume that they were of the desired type, this may not be the case.)

The pullets in Group *a* were believed to be resistant, yet when mated to susceptible and resistant cockerels, the incidence of lymphomatosis in the progeny was 11.9 and 10.4 per cent. respectively. The individual pullets in the group,

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TABLE I.—RESULTS OF MATING THE VARIOUS GROUPS OF PULLETS AND COCKERELS.

Breed	Group of pullets	No. of cockerel	No. of progeny	Mortality, excluding lymphomatosis. (*a*) Under 10 weeks; (*b*) over 10 weeks		Lymphomatosis. No. of cases. Excluding "eye" cases	Including "eye" cases	Percentage. Excluding "eye" cases		Including "eye" cases	
R.I.R.×W.L.	A (i) (17)	711	84	30 (*a*)	15 (*b*)	8	10	9.5	(14.8)	11.9	(18.5)
W.D.	A (i) (17)	203	124	21 (*a*)	30 (*b*)	9	13	7.2	(8.7)	10.4	(12.6)
W.L.	A (ii) (13)	364	66	14 (*a*)	12 (*b*)	5	7	7.6	(9.6)	10.6	(13.4)
R.I.R.×W.L.	B (10)	712	100	10 (*a*)	25 (*b*)	32	40	32.0	(35.5)	40.0	(44.4)
W.L.×W.W.	B (10)	364	74	6 (*a*)	8 (*b*)	24	30	32-4	(35.3)	40.5	(44.1)
W.L.×R.I.R.	C (8)	203	150	39 (*a*)	32 (*b*)	11	14	7.3	(10.0)	9.3	(11.6)
R.I.R.	C (8)	711	28	17 (*a*)	2 (*b*)	2	3	7.2	(18.2)	10.7	(27.3)
R.I.R.	D (bird 212)	711	25	2 (*a*)	20 (*b*)	6	9	24.0	(26.1)	36.0	(39.1)
W.L.×R.I.R.	D (bird 212)	203	4	3 (*a*)	0 (*b*)	1	1	25.0	(100.0)	25.0	(100.0)
R.I.R.	D (bird 60)	712	20	0 (*a*)	11 (*b*)	4	6	20.0	(20.0)	30.0	(30.0)
W.L.×R.I.R.	D (bird 60)	364	11	0 (*a*)	7 (*b*)	2	3	18.2	(18.2)	27.3	(27.3)

NOTE.—The figures in brackets ( ) after the letter of the group indicates the number of pullets in the group. The figures in brackets ( ) following the percentage lymphomatosis indicate the percentage calculated on the number of birds surviving ten weeks after hatching. This note also applies to Tables II and III.

TABLE II.—RESULTS OF MATING THE VARIOUS GROUPS OF PULLETS IRRESPECTIVE OF COCKERELS USED.

Group	No. of progeny	Mortality, excluding lymphomatosis. (*a*) Under 10 weeks; (*b*) over 10 weeks		Lymphomatosis. No. of cases. Excluding "eye" cases	Including "eye" cases	Percentage. Excluding "eye" cases	Including "eye" cases
a (30)	274	55 (*a*)	57 (*b*)	24	30	8.7 (10.9)	10.9 (13.7)
b (10)	174	16 (*a*)	33 (*b*)	56	70	32.2 (35.5)	40.2 (44.4)
c (8)	178	56 (*a*)	34 (*b*)	13	17	7.3 (10.6)	9.6 (14.8)
d (2)	60	5 (*a*)	38 (*b*)	13	19	21.6 (23.6)	31.6 (34.4)

TABLE III.—RESULTS OF MATING THE VARIOUS COCKERELS IRRESPECTIVE OF PULLETS USED.

Cockerel	No. of progeny	Mortality, excluding lymphomatosis. (*a*) Under 10 weeks; (*b*) over 10 weeks		Lymphomatosis. No. of cases. Excluding "eye" cases	Including "eye" cases	Percentage. Excluding "eye" cases	Including "eye" cases
203	278	63 (*a*)	62 (*b*)	21	28	7.5 (9.7)	10.0 (13.0)
364	151	20 (*a*)	27 (*b*)	33	40	21.8 (25.2)	26.5 (30.6)
711	137	49 (*a*)	37 (*b*)	16	22	11.6 (18.1)	16.0 (25.0)
712	120	10 (*a*)	36 (*b*)	36	46	30.0 (32.7)	38.3 (41.8)

however, did not all behave alike, for while in the progeny of six no lymphomatosis occurred, the remainder produced chicks in which the incidence varied from 7.1 to 22 per cent. In group *b* all the pullets produced affected progeny, and the incidence was high, varying from 32 to 52 per cent. There seemed to be little difference irrespective of the cockerel used. Group *b* pullets were not selected because of any history of lymphomatosis, but because of eye defects.

Group *c* was specially selected because of the history of lymphomatosis, and the pullets were actually members of affected families. In this group all pullets produced affected progeny, and the incidence varied from 3.1 to 20 per cent., yet all pullets in the group have died, and in two lymphomatosis has been diagnosed.

The two pullets in Group *d* (sisters), bred from a pullet which had produced affected progeny, both produced progeny of a high incidence.

Table IV shows the records of individual pullets in connection with lymphomatosis in their progeny, and in Table V there is shown a record of the tissue in which lesions were found.

From these results it seems clear that the individual pullet used for breeding is an important factor in the occurrence of fowl paralysis (lymphomatosis), and that though birds may have the same history throughout their lives and breeding, the occurrence of fowl paralysis (lymphomatosis) in their progeny may be entirely different.

Breeding in 1935.

Breeding experiments were continued in 1935. The surviving members of the 1934 groups of pullets were again bred either to the same cockerels as formerly used or to cockerels with a similar history. We have also bred from some of

the progeny of the 1934 groups, including those from families either in which there was no evidence of lymphomatosis or in which the condition was diagnosed. It is yet too early to record final results, but the position at present is that there has been fewer cases than in the 1934 groups, and there is as yet little significant difference in the incidence in the progeny of the selected pullets. Though we worked with as large a number of birds as possible, the numbers in some groups are so small that only a limited value can be attached to the percentage figures.

TABLE IV.—RECORDS OF INDIVIDUAL PULLETS SHOWING INCIDENCE OF LYMPHOMATOSIS IN PROGENY.

(Only pullets producing more than a total of 8 chicks are considered.)

GROUP A.

Pullet	Cockerel	No. of Progeny	Mortality in Progeny, excluding lymphomatosis. Under 10 wks.	Over 10 wks.	Lymphomatosis No. of cases	Percentage		Total percentage
514+	203	5	0	2	0	0	(0)	7.1
	711	9	1	2	1	11.1	(12.5)	(7.6)
524+	203	16	1	4	6	37.5	(40.0)	31.6
	711	3	0	1	0	0	(0)	(33.3)
522	203	7	0	1	2	28.5	(28.5)	14.3
	711	14	2	5	1	7.1	(8.3)	(15.7)
525+L	203	14	1	5	0	0	(0)	0
	711	1	0	0	0	0	(0)	(0)
504+L	203	17	2	11	2	11.8	(13.3)	10.3
	711	12	8	2	1	8.3	(25.0)	(15.7)
527+	203	3	1	0	0	0	(0)	0
	711	10	5	2	0	0	(0)	(0)
546+L	203	5	1	0	1	20.0	(25.0)	22.0
	711	4	2	0	1	25.0	(50.0)	(33.3)
536+	203	7	6	0	0	0	(1)	0
	711	9	4	4	0	0	(0)	(0)
540+	203	14	1	2	0	0	(0)	0
	711	0	0	0	0	0	(0)	(0)
545	203	16	2	3	0	0	(0)	0
	711	3	0	0	0	0	(0)	(0)
532	364	18	3	5	0	0	(0)	
547	364	16	5	3	3	18.7	(27.3)	

GROUP B.

Pullet	Cockerel	No. of Progeny	Mortality in Progeny, excluding lymphomatosis. Under 10 wks.	Over 10 wks.	Lymphomatosis. No. of cases	Percentage		Total percentage
475+	712	20	2	4	11	55.0	(61.1)	41.4
	364	9	1	1	1	11.0	(12.5)	(46.1)
837+	712	19	0	8	7	36.8	(36.8)	40.8
	364	8	0	1	4	50.0	(50.0)	(40.8)
109+	712	9	1	2	5	55.5	(62.5)	32.0
	364	16	0	4	3	18.6	(18.6)	(33.3)
1+	712	3	0	2	1	33.3	(33.3)	44.4
	364	6	0	3	3	50.0	(50.0)	(44.4)
385+	712	12	1	0	5	41.6	(45.4)	42.1
	364	7	1	1	3	42.8	(50.0)	(47.0)
459+	712	16	1	1	8	50.0	(53.3)	52.0
	364	9	1	4	5	55.5	(62.5)	(56.5)
762	712	14	1	4	5	35.7	(38.4)	47.4
	364	5	1	0	4	80.0	(100.0)	(52.9)

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GROUP C.

Pullet	Cockerel	No. of Progeny	Mortality in Progeny, excluding lymphomatosis. Under 10 wks.	Over 10 wks.	Lymphomatosis. No. of cases	Percentage	Total percentage
722+	711	7	4	1	0	0. (0)	8.3
	203	17	5	2	2	11.8 (14.3)	(13.3)
723+L	711	0	0	0	0	0 (0)	15.4
	203	13	2	1	2	15.4 (18.2)	(18.2)
727+L	711	14	9	0	1	7.2 (20.0)	20.0
	203	16	5	5	5	31.2 (45.5)	(37.5)
728+	711	3	0	2	0	0 (0)	3.1
	203	29	8	2	1	3.4 (4.8)	(4.2)
730+	711	1	0	0	0	0 (0)	3.3
	203	29	5	9	1	3.4 (4.2)	(4.0)
731+	711	3	2	0	0	0 (0)	3.1
	203	29	7	8	1	3.4 (4.5)	(4.3)
739+	711	0	0	0	0	0 (0)	5.3
	203	19	7	4	1	5.3 (8.3)	(8.3)

GROUP D.

Pullet	Cockerel	No. of Progeny	Mortality in Progeny, excluding lymphomatosis. Under 10 wks.	Over 10 wks.	Lymphomatosis. No. of cases	Percentage	Total percentage
212	711	25	2	13	9	36.0 (39.1)	34.5
	203	4	0	3	1	25.0 (25.0)	(37.1)
60+	712	20	0	6	6	30.0 (30.0)	28.7
	364	11	0	3	3	27.3 (27.3)	(28.7)

NOTE.— + = Died during the year. +L = Died and showed lesions of lymphomatosis. Figures in brackets ( ) in connection with the percentage lymphomatosis refers to percentage calculated on the number of birds surviving 10 weeks after hatching.

TABLE V.—RECORDS OF INDIVIDUAL PULLETS SHOWING TISSUES OF PROGENY IN WHICH LESIONS OF LYMPHOMATOSIS WERE FOUND.

GROUP A.

Pullet	Cockerel	Lesions in Progeny. Nerve tissue only	Other tissues, including eyes	Total
514+	203	*Nil*	*Nil*	0
	711	1 brachial	—	1
524+	203	1 brachial	3 heart muscle ; 1 heart, skin and ovary ; 1 iris	6
	711	*Nil*	*Nil*	0
522	203	—	1 liver ; 1 iris and choroid	2
	711	1 brachial, crural and sciatic	—	1
525+L (liver and kidneys)	203	*Nil*	*Nil*	0
	711	*Nil*	*Nil*	0
504+	203	—	1 kidney ; 1 iris and skin	2
	711	—	1 liver, kidney and ovary	1
527+	203	*Nil*	*Nil*	0
	711	*Nil*	*Nil*	0
546+L (nerves and ovary)	203	—	1 testes	1
	711	—	1 mesentery and pancreas	1
536+	203	*Nil*	*Nil*	0
	711	*Nil*	*Nil*	0
540+	203	*Nil*	*Nil*	0
	711	*Nil*	*Nil*	0
545	203	*Nil*	*Nil*	0
	711	*Nil*	*Nil*	0
532	364	*Nil*	*Nil*	0
547	364	2 lumbo-sacral	1 spleen	3

GROUP B.

Pullet	Cockerel	Lesions in Progeny. Nerve tissue only	Other tissues, including eyes	Total
475+	712	2 brachial ; 1 lumbo-sacral ; 2 brain	2 skin ; 1 ovary ; 1 liver ; 1 iris ; 1 iris and choroid	11
	364	1 brachial	—	1
837+L (iris)	712	2 brachial ; 2 lumbo-sacral ; 1 sciatic and brachial	1 liver, kidneys and lungs ; 1 iris	7
	364	1 brachial (tumour) ; 1 brain	1 ovary ; 1 sympathetic and heart	4
109+	712	1 brain ; 3 ischiatic and lumbar	1 iris	5
	364	2 brachial ; 1 sympathetic	—	3
1+	712	—	1 iris	1
	364	1 brachial, lumbo-sacral and skin	1 liver, spleen, kidney, and testes ; 1 iris	3
385+	712	1 brachial and lumbo-sacral ; 1 sympathetic	1 liver ; 1 kidneys ; 1 iris and ciliary body	5
	364	2 brachial	1 iris and choroid	3
459+	712	1 lumbo-sacral ; 1 sympathetic ; brachial and sciatic ; 1 lumbo-sacral and heart muscle	3 liver ; 1 liver and spleen ; 1 iris	8
	364	1 brachial ; 1 sciatic and ovary ; 1 brachial, sciatic and ovary	1 iris; 1 ovary, skin; 1 kidneys and spleen	5
762+	712	1 all major nerves ; 1 lumbo-sacral	2 iris and choroid ; 1 ovary, pancreas, mesentery and kidney	5
	364	2 brachial	1 ovary ; 1 ovary and mesentery	4

GROUP C.

Pullet	Cockerel	Lesions in Progeny. Nerve tissue only	Other tissues, including eyes	Total
722+	711	*Nil*	*Nil*	0
	203	1 brain ; 1 sciatic nerves, brachial heart and ovary	—	2
723+L (liver, mesentery)	711	*Nil*	*Nil*	0
	203	—	1 ovary, kidneys, heart ; 1 ovary, kidneys, liver	2
727+L (spleen, ovary, skin)	711	1 lumbo-sacral and intestine	—	1
	203	—	1 liver ; 1 testes ; 2 iris ; 1 iris and choroid	5
728+	711	*Nil*	*Nil*	0
	203	—	1 intestine	1
730+	711	*Nil*	*Nil*	0
	203	1 crural nerves	—	1
731+	711	*Nil*	—	0
	203	—	1 skin	1
739+	711	*Nil*	—	0
	203	—	1 oviduct	1

GROUP D.

Pullet	Cockerel	Lesions in Progeny. Nerve tissue only	Other tissues, including eyes	Total
212	711	—	2 liver ; 1 kidneys ; 2 iris ; 1 choroid ; 1 pancreas, lung, liver and testes ; 1 sympathetic and gizzard ; 1 sympathetic and mesentery	9
	203	1 sympathetic, brachial and kidneys	—	1
60+	712	1 brain ; 1 brachial and lumbo-sacral	1 liver ; 2 iris ; 1 liver and kidneys	6
	364	1 brachial	1 iris : 1 ovary and kidneys	3

NOTE.— + = Died. +L = Died and showed lesions of lymphomatosis. Tissues affected shown below number of pullet.

Conclusions and Summary.

As a result of the above experiments and observations, we draw the following conclusions :

(1) Lesions of lymphomatosis may be found in practically all tissues, including those of the nervous system. Lesions may occur only in the nervous tissues or in other tissues, or in both in the same bird.

(2) Intestinal parasitic infestation plays no essential part in the causation of lymphomatosis in fowls.

(3) Clinical and histological evidence of lymphomatosis may be found in fowls at ages varying from two to over thirty-eight months. In the older affected birds there is evidence that the condition did not occur at an earlier age.

(4) There is some evidence that the injection of tissues from affected birds into healthy normal stock may play a part in transmitting lymphomatosis. Much seems to depend on the type of bird injected ; some strains appear to be more readily susceptible than others. In some birds the injection of tissues from healthy birds seems to be followed by the occurrence of lymphomatosis.

(5) There is no evidence that lymphomatosis is transmitted by contact of potentially affected chicks or affected adults with healthy young chicks.

(6) Breeding experiments carried out in 1934 lead us to conclude that : (*a*) Incubation and rearing play no part in the causation of lymphomatosis, for all hatches were treated similarly, and the incidence in them did not differ materially. (*b*) The four cockerels we used with the fifty pullets seemed to play no significant part in the resulting incidence in their progeny. (*c*) Groups of pullets selected for apparent " susceptibility " and " resistance " to lymphomatosis failed to furnish clear evidence of these qualities in their progeny. (*d*) Individual pullets seemed to have a considerable amount of influence in the fate of their progeny. (*e*) Pullets in whose ancestors lymphomatosis has existed to a marked degree do not necessarily produce affected progeny. (*f*) Pullets in whose ancestors there is no evidence of lymphomatosis may produce progeny in which the condition occurs to a marked extent. (*g*) Infected dams do not always produce affected progeny. (*h*) Sister pullets (" resistant " or " susceptible ") do not necessarily all behave alike in the production of affected or healthy progeny. (*i*) Mating of father and daughter or brother and sister is not followed by a greater incidence of lymphomatosis than when the birds are crossed. (*j*) Pullets producing most cases of the disease in their offspring have remained healthy, and birds in all stages have failed to transmit the disease. These findings are obviously of a limited value, but suggest that a typical virus is not the cause of the condition. (*k*) Lymphomatosis seems to be a condition associated with individual birds.

(7) Experiments on lymphomatosis must always be observed over a lengthy period, for we have shown that birds may develop symptoms and lesions at all ages, even over three years old.

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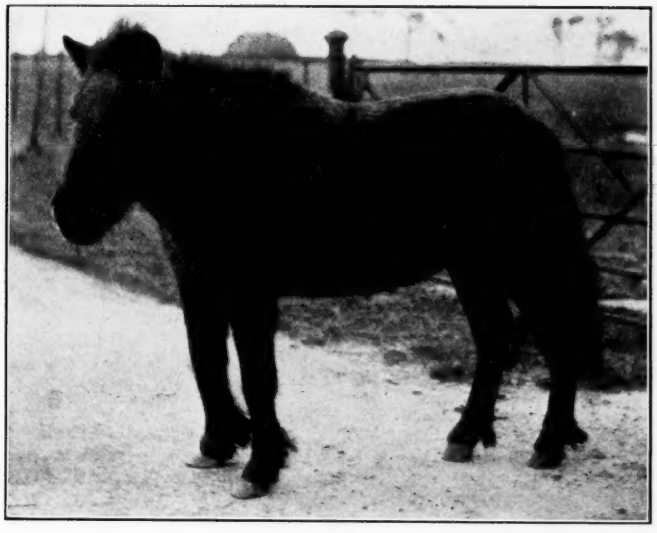

Shetland Pony aged 50 years.

(*Article by* Sir Frederick Hobday, F.R.C.V.S., *page* 331)

Avian Embryonal Nephroma.

(*Article by* H. P. Hamilton, B.V.Sc. Toronto, *page* 321)

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It is probable that heavy culling of birds carried out by experienced farmers accounts for the low incidence on some farms, and we suggest that future work on lymphomatosis should include a study of the types of birds from which to breed in order to prevent its occurrence in the progeny.

The important points from the poultry breeder's point of view are: (*a*) Are survivors in families in which lymphomatosis is present the most resistant birds? or (*b*) Should poultry farmers always aim at obtaining stock for breeding purposes in which there is no apparent history of the condition for several generations?

Such work must of necessity be carried out on a very large scale.

AVIAN EMBRYONAL NEPHROMA.

By H. P. HAMILTON, B.V.Sc. (*Toronto*).

Director of the Poultry Pathological Research Laboratory, Goring, Reading.

THE somewhat infrequent occurrence in fowls of neoplasms arising from the undifferentiated nephrogenic cells lends interest to a case encountered at these laboratories.

The tumour was found in a young Light Sussex pullet about fourteen weeks old, and involved the anterior lobe of the right kidney. It weighed 140 grammes, and occupied practically the whole of the abdominal cavity. The bird had been killed for post-mortem examination as it was in a poor condition, but no specific symptoms had been noted by the owner. The tumour was at first thought to be sarcomatous on account of the proportions it had assumed in so young a bird. Macroscopically, the tumour appeared multi-lobulated and was circumscribed by a very thin capsule. The histological picture, however, was that of a typical embryonal nephroma, the spherical cells forming irregular adenomatous structures with an attempt at glomerular formation, but which were seen to be merely a conglomeration of epithelial-like cells and fibrous stroma.

The histogenesis of these tumours is somewhat uncertain, but they are generally presumed to develop as a result of some congenital mishap. Feldman[1] states that an occasional tumour of this kind is found entirely separated from the kidney, and its histogenesis may be explained on the basis of so-called fœtal rests derived from the remains of the Wolffian body. These fœtal rests probably result from the failure of certain portions of the mesonephron to be utilised in the development of the permanent kidney. Instead of undergoing retrogression, the unused cells continue to grow, but without the guiding influences of normal tissues, and an unrestrained focus of immature elements with neoplastic possibilities results.

Matthews[2] gives the incidence of these tumours as 12 in 2,000, or 0.6 per cent. It is of interest to note the case quoted is the first we have encountered in some 3,000 tumours examined histologically. Ten of Matthew's tumours were obtained from the Barred Rock breed, and he is inclined to believe this breed showed parti-

cular predisposition to the development of neoplasms. The comparative rarity of the Barred Rock in this country affords a tentative explanation of the low incidence of this type of tumour.

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THOUGHTS—HYPOTHETICAL—ON THE THERAPEUSIS AND PATHOGENESIS OF MILK FEVER.

By W. M. SCOTT, F.R.C.V.S., Dr. Med. Vet. (*Zurich*)

Bridgwater.

Introduction.

WHEN the editor asked me to send a contribution to THE VETERINARY JOURNAL I was confronted with the difficult task of choosing a suitable subject out of the plethora available. A chain of circumstances following the request immediately arose which simplified matters and decided for me.

I was requested by a new client to attend a case of milk fever, but I was on no account to come prepared "to inject under the skin" as he had lost two cows by this method. The following day I was informed by another that he had a cow which had been treated in a similar manner, but which had developed a swelling at the seat of injection and a larger one pre-sternum: from the latter situation I took away a slough 1½ lb. in weight, the owner, too, saying, "No more skin injections for me." These stated facts urged me to review some salient points in regard to milk fever and embody them in this article.

History.—Since 1897, the year Schmidt made his epoch-making discovery, milk fever, so called, has been robbed of its sting. I often wonder if it is placed on record what logical and mental workings he adopted on his voyage of discovery, or did he just stumble upon the method of udder inflation by what in his ignorance man is wont to call *chance?* As a clinician, Schmidt gave us the remedy for milk fever, but he did not solve the *causa causens*—another illustration of practice preceding science; and it was left to two scientific researchers, Dryerre and Gregg, to find us the key to at least a part, if not the whole, solution of the pathological workings of milk fever. These workers assert milk fever is due to calcium deficiency, to remedy which they advise the injection of calcium in its most soluble form.

Deductions.—I think everyone will agree, both laboratory and field workers, that in every case of milk fever there is a calcium deficiency, and if this deficiency is made good the phenomena so characteristic of milk fever will disappear and the animal will return to normal in a large percentage of cases. Nevertheless, as one who has given the calcium treatment and the inflation treatment an extensive

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trial, I am definitely in favour of the latter; and, given a case which is taken in reasonable time, it is an almost 100 per cent. certain cure. Further, it is more certain and less erratic than the calcium method. One must, however, eliminate those cases which have been down all night showing delirium alternated with coma, every reflex absent, with cerebral effusion and maybe rupture of the capillaries of the *rete mirabilæ*. Cases such as these are of course futile from the onset. The advocates of the calcium treatment condemn inflation for its mastitis complications and consequent loss of milk. I may at once say, however, these sequelæ have given me little anxiety. My practice is to inject 250 to 300 c.c. of sterile medicated water into each quarter, and then pump in air to *tensity* point. This is important, for the therapeutics of udder inflation to my mind is largely if not purely mechanical. In the gravid uterus there is a large volume of blood, which increases with advancing pregnancy. As soon as the calf is born a recoiling uterus means a redistribution of blood, and the mammary gland claims priority for hyper-milk production. The udder becomes very active, and an excess of calcium leaves the system through the first milk. Inflation places the udder in a state of passivity, and lactiferous cell proliferation is strangled by pressure pure and simple, but the maximum pressure is obtained by liquid and air and not by air alone, which is too volatile, as I have pointed out.

It may be taken for granted, I think, that inflation stems calcium depletion, and it is conceivable that it may also be the means of driving back into the system such calcium that may be present in the udder. Believing that this hypothesis is a sound one, I never remove milk prior to inflation; but it cannot raise the content further by its own action. How, then, is it that udder inflation produces such spectacular results if calcium deficiency is the sole factor? An animal almost dead to the world now is, in a few hours, up, walking about, ruminating, and with every appearance of perfect health.

I believe the pathogenesis of milk fever is not so simple as to be solved by saying that it is due to a deficient calcium content. I feel sure there are other complex factors at work.

Let us look at the milk analysis of the human female, the mare, and the cow by way of comparison. The two former seldom or never suffer from milk fever; the latter, thanks to the fact that man has reduced the cow to a milking machine, often.

	Woman	Mare	Cow
Proteins	1.5	1.9	3.3
Fats	3.5	1.4	3.6
Milk sugar	6.5	6.3	4.8
Salts	0.2	0.4	0.7
Water	88.3	90.0	87.3

From the above table it will be seen that *proteins* are greatly in excess in the cow.

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Does this rich protein factor hold a contributory cause to milk fever? I wonder!

The three proteins in milk are lactalbumin, lactoglobulin and caseinogen, and the most important is the latter, for it contains nearly all the amino-acids which enter into the composition of the various proteins. During the metabolic changes that are going on in the active mammary gland, is there developed an active protein factor, ferment or enzyme of a volatile character which reaches the blood or lymph stream and acts upon the endocrine glands, developing a vicious circle, and culminating in milk fever? This hypothesis is rather borne out by an interesting fact I have noticed for years, *i.e.*, soon after udder inflation I invariably observe the patient becomes worse, *i.e.*, if she is on her legs her hocks straighten more, and consequently she staggers more; if in a coma, the coma

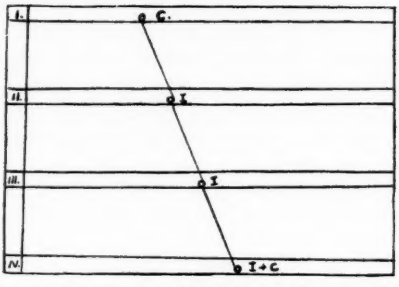

Chart showing the Four Stages of Milk Fever plus suggested treatment.
C=Calcium. I=Inflation. I+C=Inflation and Calcium.

becomes more profound. Is this due to charging the system with toxic proteins under pressure from the inflation, or is it due to an alteration in the blood pressure? *Apropos* to this, some years ago I had a case in which I had just finished udder inflation when the patient's limbs straightened, muscles became rigid, the eyeballs fixed, joints locked. This tetanised convulsion was followed by diffused tremors, then relaxation and death. Post-mortem revealed hæmorrhage at the base of the brain.

The question now arises: Can we apply the conclusions, based, as some of them are admittedly, on a hypothetical foundation, to routine practice, in the hope of bringing about a still higher percentage of recoveries in these cases of milk fever?

While placing my larger confidence every time on mammary inflation, I do think the calcium treatment in many cases is justified by results, and where one is dealing with a case in the initial stage one may prescribe this method with confidence. In the semi-advanced stage I would not gamble on the calcium method alone, and in the advanced stages most certainly not. Here I should inflate the udder to the extreme, and await results, and/or fortify my position further by the use of calcium as an *adjuvant*.

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In practice one may divide the cycle of milk fever, speaking generally, into four heads: (1) Inco-ordination; (2) paraplegia; (3) coma (semi); (4) coma (complete). According to the stage in which the practitioner finds his patient, so he must prescribe.

The foregoing chart might be useful to the younger practitioners, although, I would hasten to add, I would most strongly deprecate any hard-and-fast rule in cases of milk fever, for even here there are never two cases absolutely identical, consequently each case must needs be a law unto itself and treated on its merits.

THE POSSIBLE RELATIONSHIP OF THE PARATHYROIDS TO THE EQUINE OSTEODYSTROPHIES.

By. V BERWYN JONES, M.R.C.V.S.

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IT is only during the last decade that the controlling influence of the parathyroid glands on the maintenance of the skeletal system has been recognised. A review of the literature on this subject, from which I have freely quoted, appears to warrant a very complete research into the possible, even probable, relationship of these glands with some of the more important and crippling bone diseases of the horse.

It has recently been shown by Mitchell that most of the clinical bone diseases of the horse that we have been taught to look upon and to name as separate entities, are not localised in fact, but are part of a generalised osseous dystrophy. In no case has he found localised bone or joint diseases *per se*; always at post-mortem examinations there have been disclosed sub-clinical lesions in other bones, joints, or nerves. That such lesions can be in a very advanced condition of disease without causing apparent pain or lameness, or even loss of action, has been recently brought home to me. As a matter of interest, and as a *placebo*, I radiographed the navicular bones of a polo pony that was said to stumble very occasionally; there was no lameness or loss of action, a good foot, and no clinical evidence of disease. To my amazement both feet showed large decalcified areas in the navicular bones, whereupon the animal was destroyed, and the diagnosis fully confirmed. But it is the generalised condition which is so suggestive of a hyperfunction of the parathyroids.

It seems to be widely accepted now that in no one of the more localised bone diseases in humans does the parathyroid hormone exert any real influence. It is, of course, only reasonable to suppose that since the secretions of the endocrine glands are distributed to all parts of the body, any abnormality in their action must be a generalised action upon the tissues involved. It would be difficult to explain a selective action upon one part only of similar tissue.

The parathyroids definitely control the amount of calcium in the skeletal system by maintaining a constant Ca. Phosphate balance in the blood. If the tissues

require more calcium the parathyroids mobilise the required Ca. from the bones, that is to say that the blood Ca. concentration is maintained, if necessary, at the expense of the skeletal system. The deprivation of calcium in the bony skeleton is perfectly exemplified in Von Recklinghausen's disease (osteitis fibrosa cystica) of humans, which is always associated with one or more tumours of the parathyroid. When the enlarged parathyroid is removed, the condition, although widespread and most crippling, resolves. Pain is said to be relieved immediately after operation, and the normal calcium phosphate level in the blood stream regained, and the bone lesions slowly repair.

Always in hyperparathyroidism there is a hypercalcæmia. The stimulant for the production of the hormone—its call to work—appears to be the serum phosphatase concentration. The glands are therefore over-active when the diet is too high in Phos. and/or low in calcium. It is most interesting and noteworthy to mark that the addition of Ca. salts to diets in general does *not* necessarily lead to increased storage of lime in the bones, for an excess of Ca. will rob the body of Phos., to be excreted as Ca. Phos.; and unless extra Phos. is fed the hormone will level the balance by calling on the skeletal store of calcium. Similarly, excess of phosphorus will deprive the body of calcium.

The fact that over-activity of the gland raises the blood calcium suggested that vitamin D had a similar or synergistic action, because in each case the effects on the chemical composition of the blood and the toxic symptoms of overdosage appear similar, *e.g.*, hypercalcæmia and tetanies ; when used in therapeutic doses, however, vitamin D does not cause a hypercalcæmia such as is readily obtained by relatively small doses of parathyroid. The difference is more marked when the Ca. Phos. balance is examined, for therapeutic doses of vitamin D cause a positive balance accelerating ossification, whereas parathyroid hormone causes a negative Ca. balance, *i.e.*, a reduction of inorganic Phos. in the serum by diuresis, and excess serum Ca., with a consequent retardation of bone formation. From the evidence it would appear that the activity of vitamin D is not necessarily dependent upon the integrity of the parathyroids, in fact they appear to protect against an excess of vitamin D. It has been known for some time that vitamin D, or its stimulator, sunlight, increases the net absorption of Ca. and Phos. by the body tissues, and that lack of it produces bone disease such as rickets. In this latter, as well as in most generalised bone diseases, there is often an enlargement of the parathyroids ; this, however, is regarded as secondary—a compensatory hypertrophy.

Hypertrophy of an endocrine may be interpreted as either a sign of compensation by one gland belonging to a reciprocal system of glands, or it may be argued that such hypertrophy was brought about by the abolition of some antagonist. It appears to be certain that there is no relationship between the thyroid and parathyroid. The sum of evidence would also seem to show that the thymus and parathyroid are antagonistic , this seems a reasonable result when it is considered that the thymus does not degenerate until skeletal

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formation is completed toward adult life, and that it is not until then that more parathyroid hormone should be made available to limit calcium deposition in the bones.

The earlier observations on the possible kinships of the hypophysis are unsound, because it was not until recently that the different hormones liberated by the anterior and posterior parts were differentiated. It seems probable that excess pituitrin is antagonistic to parathyroid, and excess antuitrin synergistic, in that the blood Ca. is raised in the latter case and lowered in the former. The old idea of an antagonism of the adrenals and parathyroid is again probably at fault since the discovery that two hormones are liberated, *i.e.*, cortin and adrenalin, and the weight of evidence would tend to show that there is no physiological connection. Except that in the case of the pancreas removal of the parathyroid leads to a decreased sugar tolerance, and that parathyroid hormone augments the action of insulin, no clinical synergism has been demonstrated. With the gonads it would appear that there is no direct relationship.

It seems, therefore, that both in cases of pure or primary hyperparathyroidism and in secondary enlargements the parathyroids are not dependent on other endocrines, and that there is no real evidence of a reciprocal system of support. It would appear, therefore, that it is only the Ca. Phos. balance, or lack of balance, that exerts any marked influence on, or is influenced by, the parathyroids. The unexplained but very real influence on the nervous system, exemplified not only by the fact that a hypocalcæmia of sufficient degree will precipitate tetanic convulsions but by the fact that in the hypercalcæmia of osteitis fibrosa cystica surgical removal of the enlarged parathyroid immediately relieves the pain, offers most interesting clinical therapeutic possibilities. The injection of calcium gluconate in milk fever of cattle has already been exploited, and is of proved value. It has never been explained, however, why a lasting cure during the whole of the lactation period should result from a single injection of calcium gluconate, which it is known is eliminated from the blood stream in a few hours. The rapid and lasting cure in this disease and the immediate relief from pain and healing obtained in von Recklinghausen's disease would seem to show that although it may take a good deal in the way of what may be termed metabolic impertinences to upset the controlling balance of the glands, so soon as the poise is replaced or regained it is once more not easy to upset. From the viewpoint of the practitioner the pain factor is most interesting, and has not yet been explained. Mitchell has always found nerve degenerations in rheumatic disease of equines. If it were found that a calcium unbalance were present in such cases, it might be that either injection of Ca. or the administration of the hormone would, by correcting the balance, free the animal from pain, and allow work to be resumed.

I have been very intrigued, and perhaps encouraged, in some mild experiments that I have made in the condition known as " sore shins." Although it is early yet to make a definite statement, it would certainly appear that a timely injection

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of calcium very rapidly removes both the pain and the inflammation. I hope at a later date to report further on these cases.

I can find no trace in veterinary literature of the suggestion ever having been made that this endocrine factor could play a part in the causation of equine osteodystrophy. I would suggest that it would be most interesting, and perhaps profitable, to obtain the co-operation of a large breeder of blood stock and to determine: (1) The Ca. Phos. balance at different ages, together with the excretory quantities under a controlled diet. (2) To determine the balance in equines known to suffer from what Mitchell terms "rheumatic disease," and to include in post-mortem examinations reports of the latter disease, the macroscopical and microscopical appearances of the parathyroids. (3) To try the effect of a parathyroidectomy on a suitable case of apparently localised bone disease, such as might be represented by an early case of so-called navicular disease. To quote John Hunter's famous remark: "Why think? Why not try the experiment?"

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A CLINICAL REPORT ON THE DURATION OF LICE INFESTATION—*Linognathus Setosus*

By GEORGE DYKES, M.R.C.V.S.

Glasgow.

It might appear at first that any statement as to the duration or age of an infestation by lice (*Linognathus setosus*) would be essentially speculative. Nevertheless, as the following account of a little work done may be regarded as having some clinical and medico-legal value, I submit it with the reservation which must be placed upon a report dealing with one case only, *i.e.*, that whatever special evidence may be drawn therefrom is more of the nature of an inclusive evidence than a conclusive one of general application.

Subject.—A Cairn Terrier, female, aged seven months, was submitted for examination and opinion.

History.—The owner stated: "I bought this dog six weeks ago at a fair price. Two weeks thereafter she commenced scratching; confining her attention to the area behind the left ear. I could see no cause for the irritation, but last night a visitor to my house examined her, and drew my attention to the presence of lice. I wish you to treat her, but particularly want to know if this bitch, purchased as being in good health, was infested with lice at the time of sale?"

Although I saw that the bitch was lice-infested, I must say that I did not feel competent to venture an opinion regarding the age of the infestation, and immediate search of much literature afforded no guidance upon this moot problem, which appealed to me as of prime importance alike to the vendor and the purchaser. As much stress was to be laid upon the date of infestation, I sought to supply an approximately accurate answer which might prove acceptable to both parties. Arising from other considerations, it was assumed (*a*) that the majority, if not all, of the eggs would be laid on the hairs at or near to the skin level, and that (*b*) if the rate of growth of the hair were known, the distance between the skin surface and the most distant egg-shell should mark, within reasonable limits, the age of the infestation.

To prove the rate of hair-growth, I adopted the following steps : (1) An area of one square inch was shaved (dry) behind the left ear. The removed egg-laden hairs were retained for examination, and the animal placed under treatment. (2) On the seventh day a quarter of the above area was re-shaved. Measurements of the removed hairs showed that they had grown at the rate of 3 mm. per seven days, and so on the seventh day a tentative opinion, as hereafter outlined, was given. (3) The remaining area was similarly shaved in quarters at intervals of fourteen, twenty-eight and forty-two days. Measurements of the hairs removed from these areas at the time stated showed 6 mm., 12 mm., and 18 mm. respectively ; in other words, it was indicated that the rate of growth was at a constant rate of 3 mm. per seven days.

NOTE.—Subsequent observations on other dogs have shown that under almost similar conditions of season, domestication, etc., the rate of growth is maintained at the rate of 3 mm. per seven days consistently until the maximum length of hair peculiar to breed and area is reached.

(*b*) Examination of nits on hairs : (1) The *lowest* nits were found at slightly less than 1 mm. from the skin surface ; contents, granular. (2) The *highest* intact nits were found at 5.5 mm. from the skin surface ; these showed clearly distinguishable, well-formed larvæ of *L. setosus*. (3) Beyond 6 mm. from the skin surface up to 20 mm. empty egg-shells only were found. (4) Beyond this distance, 20 mm., there was no evidence of cementum found on the hairs.

Co-relating the evidence (*a*) that growth of hair was at the rate of 3 mm. per week, and (*b*) that the highest discoverable egg-shell found on hairs taken from what appeared to be the primary focus of infection was 20 mm. from the skin surface, I expressed the opinion that the bitch had been infested at between the sixth and seventh week prior to my examination. When this tentative diagnosis was expressed, the purchaser communicated with the vendor, who not only denied the presence of lice in his kennels, but invited me to inspect his stock. At his premises, two adult bitches and four dogs of the same litter as the subject in question were examined, with a negative result. It emerged, however, that my client's bitch had been sent on approval to a probable purchaser for three days prior to having been forwarded to my client, and that on its return it had

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not been re-admitted to the vendor's kennels, but had been despatched forthwith to the purchaser. Pursuing the matter, it was found that the person who had the dog on approval owned an aged spaniel that was heavily infested with lice (*L. setosus*).

Summary.—Bearing in mind the aforementioned reservations, this report seeks to show a means whereby an acceptable assessment of the duration of infestation by *L. setosus* may be arrived at by measuring the distance from the skin to the furthest out egg-shell on the hairs, and at once relating that measurement to the average rate of growth of hair on dogs, *viz.*, 3 mm. per week until the maximum length of hair peculiar to the breed is reached. I have no other evidence that oviposition takes place other than 1 mm. above the skin surface, and conclude that the after-position of the fixed and soon empty egg-shell may be taken as the time index as to when oviposition first occurred on a recent case.

Acknowledgments.—My grateful thanks are due to Professor Hugh Begg, F.R.C.V.S., for his interest in, and his encouragement to publish, this report. I also wish to express my indebtedness to Professor J. W. Elmslie, B.Sc. Agric., M.R.C.V.S., for the ready way in which he placed laboratory facilities at my disposal.

ON SWERVING TO THE RIGHT.

By DAVID ERIC WILKINSON, M.R.C.V.S.

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SOME year or two ago, at a meeting of the Central Veterinary Society, a paper on " Diseases of the Heart " was jointly presented by three practitioners. The writer was unfortunately prevented by a professional engagement from being present, although most anxious to attend in order to note whether any of the essayists or those taking part in the subsequent discussion would make any reference to a peculiar habit which he has for many years regarded as symptomatic of painful affections of the heart. No such reference was made, nor has the writer been able to trace any remarks on this habit in any veterinary literature.

The habit referred to here is that of swerving to the right. Most practical horsemen will agree that when a ridden horse refuses he nearly always swerves to the left, *i.e.*, away from the whip hand! The writer has noticed, however, during a lifelong experience of hacking, hunting and driving, that horses which swerve to the right under pressure or when distressed always, sooner or later, develop heart disease of a serious character.

The first occasion on which this was forcibly brought home to him was when a horse jumped a fence in a hunters' steeplechase immediately in front of his own mount and fell dead on landing. A post-mortem revealed enormous hypertrophy of the animal's heart, which weighed 38 lb. Yet this horse had been winning steeplechases for several years, his last winning race being about a week before

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he fell dead. His habit of swerving to the right when tiring at the end of his races was well known to contemporary riders, who were careful to keep on his near side if possible.

Some years ago two horses well known to the writer, one of them a previous winner of the race, were out by themselves as they approached the last fence in the Grand National. The previous winner was considered by most good judges of steeplechasing to be at least 10 lb. a better horse than the other, yet to their amazement he was beaten. The explanation offered by two independent and very competent judges who knew all the circumstances was that the rider of the beaten horse was warned by the trainer on no account to get on the off side of his opponent if they were left in the lead together, as that horse had a dangerous habit of swerving to the right when tired, and might collide with a horse on that side and knock him over. Riding to orders, the jockey, a most capable and conscientious horseman, steadied his mount in order to avoid the risk of a collision. Unfortunately for him his opponent on this occasion jumped his fence quite straight, and the other horse could not catch him on the run in.

The sequel is interesting. The winner was a strong winter favourite for the next Grand National, but was withdrawn from the race as he was found to be suffering from heart disease. He was never able to stand training again, and has not raced since.

A very high-class two-year-old showed similar tendencies to swerve to the right towards the end of his first racing season, and his three-year-old racing career was ruined by this habit. Amongst racing men he came to be described as an arrant coward who was tired of racing. Later he was found dead in his box, and a post-mortem revealed a chronic pericarditis and pleuritic adhesions on the left side of the chest.

An examination of many harness horses and heavy carthorses said to be "jibbers" has brought to light numerous instances of valvular disease and mitral regurgitation. Cross-examination of the drivers has usually elicited the information that these horses "swerved to the right" before completely stopping.

EQUINE LONGEVITY.

By SIR FREDERICK HOBDAY, F.R.C.V.S.

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THE recent report in *The Times* and other papers of the death of a forty-year-old horse at the Home of Rest for Horses, Boreham Wood, Hampshire, has raised considerable interest in the subject of longevity in the equine species, and has, incidentally, added a very fine specimen of a complete skull to the Museum of the Royal Veterinary College. The photograph herewith adds one more to the list, the pony belonging to Mrs. Marian Gibbs, Hartly, Melton Mowbray, who writes: "I thought it might interest you to know that we have a Shetland

pony named Dot whose age is fifty. She has been in our possession sixteen years, and we know nearly all her life history from the time she was sent in a big crate from Scotland to Melton Mowbray for a little boy who is now a man aged forty-eight." There is also a print in the Council Chamber of the Shire Horse Society in London of "Old Billy" who lived to be sixty-two.

TORSION OF THE UTERUS.

By RICHARD HUDSON, M.R.C.V.S.
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TORSION of the uterus is most commonly met with in cows and heifers. It has been observed in the mare, ewe, sow, bitch and cat, but its occurrence in these animals is rare. We know little about its cause. Probably most cases are due to fœtal movements, but some may be due to accidents, and in the mare to rolling. In one case with which I had to deal there was a history of the heifer having been pushed sideways into a ditch a few days before, but usually one fails to obtain any evidence of such accidents. In most cases the patients have gone to normal time, and after straining for several hours nothing has resulted.

In my cases the symptoms of obstructed parturition have not caused any alarm. There has not been acute distress, or violent efforts, but slight straining, and nothing comes forward after the usual delay. Sometimes the owner remarks that "she has been at it from twelve to forty-eight hours, but doesn't get on with it."

On examination the entrance to the uterus is found to be either partly or totally occluded. In partial torsion, after searching for an entrance, one usually finds that the fingers have to go round a fold of the vagina or os before they can enter. In complete torsion they come up against a blind end through which nothing can penetrate. There are said to have existed cases of torsion where no spiral folds of the vaginal wall have been present, but it is difficult to imagine such a case except in cows, bitches and cats, where one horn only may be twisted.

It is necessary to determine in which direction the folds of the vaginal wall turn, because it will determine the direction in which the patient is to be rolled. If, using the right hand, the fingers follow the folds upwards from left to right, the patient must be rolled in that direction, and *vice versa*.

In cases of partial torsion, where the hand can be introduced, it is no use waiting for further dilation. If it is possible to reach the head and fore feet, it is often possible to effect delivery without casting and rolling the patient, and without injury. The head should be roped, and then the feet, and traction exerted upwards by a small pulley attached to the top hinge of a door or to a roof spar. The

lifting action tends to bring the womb towards the operator, and at the same time to relax the twist. The head of the calf should be brought through first, followed by the feet, which must have been securely banded, for if a rope slips off, it is likely to be a difficult job to replace it. In one case when traction was applied to the head, the calf, being alive, gave a violent lurch, and the twist was completely removed, making delivery easy.

In complete torsion it is necessary to cast the patient, and for this I prefer sidelines with rope hobbles long enough to pass twice round the legs above the fetlocks. After securing the fore feet the ends of the rope may be passed under the back and tied to the opposite legs to bring the legs closer to the body and so make rolling easier. If the patient is in a stall, a rope may be passed under her. One end is made fast to a hind limb, and the other to a pulley fixed to the roof. When drawn on, the rope lifts and turns the cow in the space she lies on. It may be necessary to turn the animal several times before the twist is removed, and sometimes it is impossible to remove it, usually owing to tympany of the rumen or abdomen from peritonitis. If tympany of the rumen exists, it is better to puncture it before rolling. If tympany is due to peritonitis, and no reduction of the twist takes place on rolling, the sooner the animal is slaughtered the better.

If one has been successful in reducing the twist, and the os can be entered, it may be necessary to wait until it is sufficiently dilated to allow of safe removal of the calf. To force delivery through an incompletely dilated os is risky, and if it is attempted, plenty of time should be allowed during the application of gentle traction for dilatation to take place.

Cases of torsion of the uterus do not always do well, even after a satisfactory delivery. Some, if examined *per vaginam* a few days afterwards, present a well-defined swelling, often on both sides of the pelvis, and extending into the abdomen. They may have resulted from hæmorrhage into the broad ligament or peritoneum, and I think they are more likely to result from the twisting of the uterus than from any injury during delivery.

I have only seen two cases in mares. The first was dying from hæmorrhage when I arrived. She had been in labour several hours, and on my arrival was straining constantly, forcing out a quantity of blood each time. She was in a hopeless condition, and so was shot.

The second case occurred recently. She was a heavy shire mare, a month over-due to foal. She had been uneasy for several hours, lying down and showing slight pain, then immediately rising and continuing to graze about the paddock. On examining her the os could not be felt, and distinct spiral folds of the vaginal wall were present. Her general condition was good, so I returned home for casting ropes, only a short distance. When I returned I found her sweating and breathing heavily, and it was evident that something serious had happened. However, she was cast and rolled without delay. After giving half a turn over

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the twist was partly removed, and after a complete turn it was removed entirely. Unfortunately the mare did not live long enough to benefit by the operation. Had I been called earlier, or cast her on my first arrival, I might have saved her.

SALMONELLOSIS IN PALMIPEDES.

By Lieut.-Colonel DUNLOP YOUNG, O.B.E., D.V.S.M., M.R.C.V.S.
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FOOD poisoning in man is generally the result of contamination by one of the Salmonella group of bacteria, and is therefore of great importance to the public health veterinary officer; therefore the work of Lesbouyries and Berthelon on "Salmonellosis in Palmipedes," from the *Recueil de Médecine Vétérinaire*, per *Veterinary Medicine*, will be of special interest.

In a long meticulously detailed discourse on paratyphoid infections of ducks and geese, the authors begin with a highly informative history of paratyphoid organisms. The term "paratyphoid bacillus" was employed for the first time in 1896 by Achard and Bensaude to designate a bacillus closely related to *Bacillus typhosus*, now known in the literature as *Erberthella typhi*, causal organism of typhoid fever in man. Later (1900) Schottmuller, Brion, Kayser and others classed other organisms in the group, which they attempted to identify as A, B and C. A and C play no part in animal pathology, while paratyphoid bacillus B, on the contrary, is of eminent importance.

Salmonella suipestifer (*B. suipestifer*) (*B. cholera suum*), isolated by Salmon and Theobald Smith in 1886 from hogs dead of hog cholera; the bacillus of Gartner, identified in 1888 as the cause of meat poisoning in man; the bacillus causing a gastro-enteritis isolated by Noble in veal; and the *Salmonella abortivo-equinus* (*B. abortivo-equinus*) isolated by Kilborne and Smith in 1898, were the organisms that Lignières placed in the B group (*S. schottmulleri*), and for which was coined the name Salmonella. The classification of these varies somewhat with the different authors. It was not until 1931 that the International Commission of Microbiology placed paratyphoid A (*S. paratyphi*) and the bacillus of Eberth in the category. At the same time American bacteriologists classed *B. pullorum* and *S. gallinarum* (*B. typhi gallinarum*) in the Salmonella group, although the latter two do not conform to the stated requirements, as neither is mobile, nor reduces neutral red. *S. gallinarum* does not form gas in sugar media, and *B. pollorum* does not ferment maltose. Moreover, while all of the Salmonella of fowl are pathogenic for man, these avian typhoid organisms are entirely harmless for man, notwithstanding the consumption of large quantities of infected eggs.

The organisms possessing the cultural and biochemic characteristics interesting veterinarians are *Salmonella schottmulleri*, *S. enteritidis* (Gartner), *B. enteritidis breslaviensis* (Aertrycke), *S. anatum*, *S. suipestifer*, *Abortiva* (*var.* equine and ovine), and the *S. typhimurium*. Essentially the differentiation

of these is based upon reactions of sugar media. The authors carried on extensive studies to appraise the observation of Bergy, Standfuss, and others, by using various specimens and media. The results are recorded on extensive tables, including identification by means of serological tests. The latter have led to more perfect differentiations. In 1934 Standfuss pointed out that in veterinary medicine the need of a differentiation has never been presented, although Salmonella affecting fowl are of great concern in public health. They can be dangerous, toxi-infectious, and cause fatal, acute, gastro-enteritis in man through the ingestion of infected meat and eggs. Eggs, badly preserved, and meat of sick fowl are particularly dangerous.

The eggs of ducks and geese are especially infective. Improperly cooked fowl liver is most dangerous. Intoxications from pigeons and chickens are less numerous. Eggs of infected Palmipedes soiled with excrement and exposed to warm temperature are highly infective. The organisms penetrate the shell to the white and yolk of the soiled eggs. In other instances the eggs infection is of ovarian origin. Paratyphoid B survives in refrigerated eggs. From the standpoint of public health it is essential to kill and burn infected fowls. Laboratory workers become gravely infected.

Salmonellosis is acute and subacute. In ducklings and goslings the mortality runs as high as 90 per cent. to 95 per cent. It is less acute and fatal in adults. It is the " keel disease " of Rettger and Scoville. The disease may appear at the age of six days in the form of diarrhœa and extreme prostration, killing in several hours in many instances. The lesions are septicæmic alterations. The few victims that recover are carriers of the organism, and these may become suddenly stricken and die in several days or longer, showing diarrhœa and enteritis.

Diagnosis.—In addition to the clinical symptoms (diarrhœa, prostration, high mortality), fruitful bacteriological examinations are made on the living (eggs and excrement) and post-mortem from the ovaries, testicles and various organs. The egg shell is contaminated with the excrement contacted in laying, which, as stated above, enters the content of the egg itself.

Treatment.—The medicinal treatment consists of general and intestinal antisepsis. Although not effective in the acute case, it overcomes the danger of carriers to the susceptible cohabitants and to man. Vitamin E should not be lacking in the feed.

Prophylaxis.—In disease-free flocks care must be exercised in adding new fowl or progeny of infected eggs. New purchases should be serologically tested (agglutination), and only non-contaminated eggs for hatching should be accepted. Where the disease exists the carcases should be burnt, and special care exercised in the collecting and disposing of excrement. In the course of an outbreak, vaccination of the healthy fowl gives a favourable result, and, of course, thorough disinfection is not omitted.

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FOWL CHOLERA.

Pasteurella aviseptica: Behaviour and Immunization. Comparison with P. Suiseptica.

By Z. MORCOS and A. HOSNY MAHMOUD,
Department of Bacteriology, Veterinary School, Cairo University.

Literature.

(1) *Reis* found that diagnosis could be arrived at by culture from a dead fowl after burial and by animal inoculation up to the fourth day, whether the carcase was buried or still in air.

(2) *De Volt and Davis* described an outbreak in turkeys. The mortality was 17 per cent. He isolated a Gram negative organism which proved fatal to chicken in inoculation.

(3) *Klimmer* prepared the following medium to differentiate between *Pasteurella aviseptica* and *Salmonella gallinarum*, Klimmer's brom.-cresol purple medium, viz :—

Normal horse serum,	90 c.c.	Reaction pH 7, and 4 c.c. of saturated solution brom.-cresol purple are added.
15 per cent. caustic soda,	10 c.c.	
Liebig's extract,	3 grams	
Peptone,	5 grams	
Saccharose,	10 grams	*P. aviseptica* produce yellow coloration, *S. gallinarum* caused *no* change.
Agar,	20 grams, and	
Distilled water add to make	1,000 c.c.	

(4) *Skidmore* proved that flies fed on blood of dead fowl cholera cases were infective to birds eating up such flies. Infected blood on a glass slide proved infective after drying at room temperature up to the eighth day only.

(5) *Hendrickson and Hilbert* isolated *Pasteurella aviseptica* by culture four days before death, one case was recorded 49 days preceding death.

(6) *Emmel and Boevers* found that Brucella and Pasteurella are inter-agglutinable *in vivo*, but on experimental infection the anti-body producing power of *P. aviseptica* is low while that of Brucella group is high. Both infections, however, could be distinguished through bacteriological grounds.

(7) *Knoth* described an outbreak in pigs with Pasteurella following the ingestion of fowl cholera dead infected fowls by the pigs, and bipolar organisms were recovered from the dead pigs and proved fatal to mice.

(8) *Urabain and Goret* infected fowls by feeding on *Pasteurella aviseptica*. Heated cultures given *per os* did not produce immunity as the birds died on inoculation of minute doses of living *aviseptica* culture.

(9) *Trillat* proved that infection in fowl cholera could take place *via* air, more so when the atmosphere is damp.

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(10) *Acevedo* used formalised cultures of *aviseptica* with success on mice in 75 per cent. of the cases which resisted test doses.

(11) *Tomasec* prepared an attenuated vaccine through transplanting the organisms three or four times a day on bile broth and found that the virulence was lost by the seventieth culture. Such a vaccine was found non-virulent for fowls and ducks, but gave variable results in turkeys and geese, and, on the other hand, was constantly virulent for pigeons.

(12) *Kalikin* favourably reported the prophylactic and curative effects of salicylic acid when given to birds *per os*. Kalikin also used successfully salicylated cultures as antigens (100 c.c. of broth *aviseptica* culture 5 days old plus 0.25 gram salicylic acid). The average dose used was 5 c.c.

Experimental.

The organisms used, *Pasteurella aviseptica*, were recovered from dead fowls obtained at the Guiza Veterinary Quarantine, through the kind permission of the Director of the Veterinary Service, Ministry of Agriculture, Cairo.

Egyptian bred fowls and Egyptian bred pigeons were used.

Morphology and Biological Characters of P. aviseptica.—Short oval cells, average length one micron, occurring as single cells non-motile. The organisms stain readily with ordinary stains, Gram negative, *non-capsulated*, show distinct bi-polarity in the blood and on first culture; on subculturing, however, the bi-polarity disappears. No spores were seen.

Sugar Reactions.—Glucose, acid produced by all strains isolated; Maltose, *majority of strains produced acid*, some no reaction; Saccharose, acid produced by all strains; Dulcite, no change; Mannite, acid produced by all strains; Litmus milk, no change; Indol production, indol produced by all strains; Gelatin, not liquefied; Lactose, no change.

Pathogenicity.—Living broth cultures of *P. aviseptica*, 48 hours old, were used.

Experiment I.—Two adult fowls (controls) inoculated intramuscularly 0.2 c.c.; two fawn desert mice, subcutaneously 0.1 c.c.; two guinea-pigs, subcutaneously 0.2 c.c.; two rabbits, subcutaneously 0.2 c.c.; two pigeons, intramuscularly 0.1 c.c.; two white rats, subcutaneously 0.1 c.c.

Result.—All birds and animals (except guinea-pigs and white rats) were found dead next morning. Pasteurella seen in blood films and recovered on cultivation from all.

Guinea-pigs and white rats were kept under observation for one month and did *not* die; *resistant to small subcutaneous doses*. In a following experiment *Pasteurella suiseptica* proved fatal to guinea-pigs but not white rats; this, in our opinion, is a sound differentiating point between *aviseptica* and *suiseptica*.

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Immunity Against Fowl Cholera.

(*Broth cultures of P. aviseptica, 48 hours old, were used.*)

Antigen I.—Cultures heated 70° C. for 30 minutes, 0.25 per cent. carbolic acid added for preservation; subcultures, 24 hours later, produced no growth on three days' incubation. *Antigen II.*—Commercial formalin was added to make up the dilution of the culture 1 : 1,000, kept at room temperature for two days; subcultures were then negative after three days' incubation. *Antigen III.*—Exactly as in II, but carbolic acid was added to make up the dilution 1 : 100.

Experiment 2.—(*a*) *Three adult fowls* were inoculated with 1 c.c., each intramuscularly with *Antigen I;* (*b*) *three adult fowls* were inoculated with 1 c.c. each into the breast muscles with *Antigen II;* (*c*) *three adult fowls* were intramuscularly inoculated with *Antigen III*, each 1 c.c.

Test dose was injected 10 days after antigen inoculation; each fowl was inoculated with 0.2 c.c. living culture into the breast muscles; two fresh fowls (controls) were intramuscularly inoculated with the very same living culture, one given 0.1 c.c. and the other 0.2 c.c.

Result.—Controls died within 48 hours of inoculation; organisms recovered. Fowls inoculated with Antigen I (heated culture) died, one 48 hours after test dose, the second and third were found dead on the fourth day of test dose, *aviseptica* was isolated from the dead fowls. Fowls inoculated with Antigen II and Antigen III resisted test dose and were kept for 30 days under observation without showing any illness.

Pasteurella suiseptica.—While doing the above work, a swab from a dead pig was received, and fortunately *P. suiseptica* was isolated. Morphology and cultural characters were indistinguishable from *aviseptica.*

Biological Reactions.—Sugar reactions, milk fermentation and indol production as given in *aviseptica*, except that dulcite was fermented with acid production.

Experiment 3.—*P. suiseptica* broth, living cultures, 48 hours old, were used. Three fowls immune to fowl cholera, 0.3 c.c. intramuscularly each; three fresh non-immunised birds, 0.3 c.c. intramuscularly each; two guinea-pigs subcutaneously with 0.3 c.c. each; two white rats, subcutaneously with 0.2 c.c. each; two desert mice, subcutaneously with 0.1 c.c. each; two pigeons, intramuscularly, 0.3 c.c. each; two rabbits, subcutaneously, 0.3 c.c. each.

Result.—All fowls and white rats did not succumb, kept three weeks under observation. Mice died next morning, also rabbits, organisms recovered. Guinea-pigs were found dead on the morning of the fourth day, organisms recovered. The pigeons were found dead on the fifth morning of inoculation and the organisms were recovered from the blood.

Summary.

(1) Both *Pasteurella aviseptica* and *suiseptica* are morphologically and culturally indistinguishable.

Pasteurella aviseptica is highly pathogenic to fowls and practically non-

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virulent to guinea-pigs on subcutaneous inoculation, while *suiseptica* is not pathogenic to fowls and is virulent to guinea-pigs.

(2) Formalin or carbolic acid on addition to *aviseptica* broth cultures do not interfere with vaccine, while heating seems to interfere with the antigenic properties.

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Clinical Articles

A NOTE ON RABIES.

By S. P. DESHPANDE, G.B.V.S.

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RABIES is a very common and most unfortunate disease in veterinary practice, especially in dogs. No attempt at treatment or relief can be made. Besides, it is not advisable to do so. If anything in the form of narcotics is given it is likely to mask the characteristic symptoms and make diagnosis more difficult. Either the patient is allowed to die a natural death or be destroyed when diagnosis is established beyond doubt.

In this article the writer wishes to record certain symptoms of rabies which one comes across in daily practice. The symptoms of rabies, especially that of dumb form, are varied, and are likely to escape one's notice unless one has extensive practical experience.

There being no legislation about the control of stray dogs, this disease is taking a heavy toll of dogs and cattle in this country. Rabid cattle are not generally brought to hospital. It is mainly in dogs—which are pet animals, and are generally brought to hospitals, especially in cities—that this disease is commonly observed. It is really a great menace to the dog world, and in turn to human beings too. Costly and well-bred dogs stand a great chance of getting infection in spite of every precaution taken by the owner. For instance, a dog is being taken on a lead for a walk ; some stray dog suddenly appears and bites it, and then disappears, keeping the owner in doubt as to what type of dog it was—rabid or normal. We have had many such cases.

At this hospital we have seen cases of rabies in horses, cows, buffaloes, sheep and goats, and dogs ; in the last, we get a number of cases all the year round.

There are two forms of this disease, *viz.*, furious and dumb. The furious form is very easy to detect, and the symptoms are clear, *viz.*, changed appearance, vacant expression, very red eyes, and the animal rushing at every object which comes into view. Such dogs do a great deal of damage by biting persons and animals, and thus spreading infection. In cases of the furious form the writer has observed that in the history given the dog is usually said to disappear from the owner's house for two or three days, and to have come back with the symptoms described above, and in a most exhausted condition, biting everything that it comes across in the compound. In this form, dogs are seen eating their own fæces. If allowed to live, these furious symptoms are followed by exhaustion, paralysis and death. Many cattle are infected by dogs suffering from this form of rabies. The symptoms they show are as follows :

In the dumb form a variety of symptoms is observed. The melancholy stage is common to both forms, and it may readily be passed unnoticed, duration being short, and also being not in any way very diagnostic in the first instance ; but when the disease advances, pronounced symptoms are shown as follows :

Uneasiness of a very peculiar type, congestion of the conjunctivæ, salivation from the mouth, drooping of the head, changed behaviour, paralysis of the lower jaw, tongue of a leaden colour, vague and listless expression, and inco-ordination of the hind limbs, which afterwards develops into complete paralysis. The eyes are sunken in their sockets, and the animal is prostrate, breathing very heavily. In the next stage the patient gets convulsions, breathing becomes more distressing and stertorous, and the dog becomes exhausted and dies.

Wasting of muscles is very rapid in rabies ; so much so that the dog may become a living skeleton within a few days. In many cases appetite is totally suspended ; but in some the animal tries to lap and eat up to the last. In some cases there is simply dry retching of a peculiar type, and uneasiness is noticed. The animal retches off and on, and a peculiar noise is produced. In some cases there is simply a vague expression and a slight change in behaviour, which are noticed for four or five or even more days, and then sudden death takes place with convulsions. The period of convulsions may last for a few hours. Another

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symptom which is seen in the case of males is that the dog frequently gets erection of the penis and makes actions as if in coitus incessantly, until paralysis supervenes and death ensues. This symptom is accompanied by a curious vague expression of the countenance.

On rare occasions the disease develops as a complication to another disease, and this is common with distemper. The only explanation for such an occurrence is that the patient must have been pre-infected, and this disease comes out as a matter of loss of vitality or predisposition due to the original disease. We had one case with typical symptoms of distemper to begin with which was treated as an out-patient for some days. After some days' treatment the patient showed symptoms of rabies ; it died, and upon the brain being sent for microscopical examination this diagnosis was confirmed. Temperature in almost all cases is elevated, in some cases up to 106° F. In some cases simply uneasiness of such a severe type is noticed that the animal does not rest in one position even for a few seconds. Such a case is very acute, and death is very sudden, often within twenty-four hours.

In the case of dogs, males are affected more than the females, probably on account of the fact that the former are fighters and more likely to be exposed to infection from this cause. Very small puppies—even two months old—are also affected. We have seen two cases of furious rabies in small pups.

In the case of cattle, it is observed that the disease frequently occurs just after calving.

Also the place and depth of bite wound are points worth considering. The deeper the bite and the nearer to the brain, the greater the possibility of developing the disease. We have seen cases bitten on the nose and head to develop the disease within twenty-five days from the date of bite. Superficial bites elsewhere on the body have remote chance of getting infection. Also the quantity of poison injected is a factor worth considering. Unfortunately this cannot be estimated.

Before concluding, it would not be out of place to mention some diseases which show at times the same symptoms as that of rabies, and are likely to be mistaken for rabies *prima facie.*

(1) Presence of a bone in the throat ; dry retching, salivation and uneasiness.

(2) Acute rheumatism ; uneasiness, peculiar gait and the animal snapping on palpation.

(3) Advanced cases of canine typhus, mouth held open, due to ulceration of buccal membrane, salivation, and exhaustion.

(4) Milk apoplexy : uneasiness, dulness, gasping (due to high temperature), and suddenness of attack.

(5) Ordinary convulsions of epilepsy when the animal is brought to hospital in the last stage and without history.

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(6) Very severe form of skin diseases. Irritation of the skin produces sometimes the same train of symptoms.

(7) Severe form of canker, making the dog uneasy and to run with the head held low and flapping of ears. Even salivation is seen.

(8) Motor or cycle accident cases with no fracture, but simply a smash; their wavering gait, salivation and dulness.

(9) Nervous form of distemper.

(10) Dislocation or paralysis of the lower jaw.

(11) Vicious temperament.

(12) Phosphorus or strychnine poisoning.

Reviews

Veterinary Posology. By G. A. BANHAM and W. J. YOUNG. Sixth edition. London: Baillière, Tindall & Cox. Price 8*s*. 6*d*. net.

WHEN a book has passed to its sixth edition, especially in a small profession like ours, it requires little else but this fact alone to recommend it as being a book which should have its place, not on the bookshelf of every veterinary surgeon's library, but on his desk, or even in his pocket, where it can be referred to at a moment's notice. Banham's " Posology " has been for nearly fifty years the little pocket companion of every veterinary student, and when he has graduated this little volume has been one of his most treasured books of reference. It has been again brought up to date, and as veterinary science has progressed its size has naturally increased also. This is the only fault we have to find with it, as it has now become a text-book, and to carry it always in one's pocket is not universally convenient. The added authorship of Mr. Jackson Young has been a wise one, and the book is thoroughly brought up to date, with chapters on the latest sera and vaccines, the application of the latest anæsthetics, the latest microscopic stains and methods, means of identification of mange parasites, details for intradermal and of the other tests for tuberculosis and glanders, and even a paragraph of such legal measures as the Horse-Breeding Act of 1918 and the Animals' Anæsthetic Act of the following year. It is, indeed, a compendium of usefulness, and should be kept, as already stated, close at hand for ready reference.

Handbook for Veterinary Surgeons. Third edition. By FRED BULLOCK, LL.D. Pp. vii + 302. London: Baillière, Tindall & Cox. Price 7*s*. 6*d*. net.

THIS new edition of Dr. Bullock's handbook has been revised and brought up to date, and in many parts entirely re-written. It is an authoritative treatise on the law governing veterinary practice and should therefore be in the hands of every student as well as every practising veterinary surgeon. The chapter on professional conduct sets out and explains in detail the by-laws of the R.C.V.S., and gives answers to all the questions which arise in regard to such important details as announcing changes of address, the running of boarding kennels, the use of signs and name-plates, the carrying on of branch practices and places of call, the employment of assistants and locums.

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It also explains the rules as to advertising, touting, covering, consultations, etc. The general advice to new graduates will be found most helpful, especially in regard to the purchase of a practice or a partnership. In two important chapters the legal responsibilities of veterinary surgeons are described under the heads of " Common Law Duties," and " Statutory Duties." The duty of care, the liability for negligence, the law as to the issue of certificates, the boarding of animals, the responsibility of the practitioner for correct diagnosis, and care in the performance of operations, are among the headings dealt with. A new section dealing with the law of contract, including the difficult questions of warranties and livestock insurance, has been introduced, which will be of the greatest value to young graduates. The chapter on the duties of veterinary surgeons under Acts of Parliament and Statutory Orders is a complete summary of those to be observed by general practitioners. The handbook is, however, not designed to be a complete treatise of the duties of whole-time veterinary officers. The advice given in the excellent chapter on the " Veterinary Surgeon in the Witness Box " is invaluable, and if followed would greatly enhance the reputation of the profession. The chapter on "Taxes and Insurance" is worth reading by everybody—even by the oldest established practitioner—for the hints given on preparing income-tax returns will show how much may be legitimately deducted for the expenses of running a practice. The book, small though it may be in actual size, with its carefully and well-arranged range of subjects, is a veritable reference dictionary of essential veterinary matters, which appeals equally to the student for his professional examination in jurisprudence and to both the young and the older graduate as a guiding-rein alongside which he must run if he desires to keep free of professional pitfalls whilst practising his profession—no matter to which branch he has chosen to become attached.

News

PRODUCTION OF FAT LAMBS.

Some interesting work has been done on sheep crossing for lamb production by the Yorkshire Council for Agricultural Education of the Leeds University at their farm, Askham, Bryan, York. The numbers reared, expressed as percentage of numbers born, were as follows: Southdown, 92.1; Suffolk, 88.3; Ryeland, 86.7; Shropshire, 86.0. The Suffolk cross, with an average of 1.63 lambs born per ewe, proved to be the most prolific. The mortality was lowest in the Southdown cross. There was most trouble at the time of lambing with the Ryelands and Shropshire crosses, and the number of deaths during the period between lambing and time for selling was greatest in the Suffolk crosses. The Southdown crosses were quite definitely fat at lighter weights than the others. Ryelands were a good second. The conclusions arrived at were satisfactory. Fat lambs for sale from the middle of June onwards can be produced from Cheviot ewes when crossed with Ryeland, Shropshire, Southdown or Suffolk rams. The financial returns showed Suffolk first, Southdown second, with Ryelands and Shropshire equal as third.

TUBERCULOSIS IN U.S.A.

Tuberculosis in Illinois causes mortality of 42.2 per 100,000 in the white population and 257 per 100,000 in the coloured. The State of Iowa has now been declared

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free from tuberculosis in cattle. Connecticut is the latest addition to the list of States practically free. This brings the number of States up to 39, leaving only nine in the non-accredited list.

According to Capt. B. Tay, M.R.C.V.S., there are 39,656 horses in Greater London.

Vaccination against Bang's disease is being inaugurated by the Federal Bureau of Animal Industry, U.S.A.

The Stoke-on-Trent Veterinary Officer has been instructed by the Corporation to arrange for the abattoirs to be closed on Sundays.

The Australian Meat Board announces the gift of £3,500 to the Council for Scientific and Industrial Research to assist experiments in chilling out and investigations of other aspects of meat export.

The R.A.V.C. Old Comrades' Association Annual Dinner will take place at Anderton's Hotel, Fleet Street, London, E.C. 4, on Friday, October 2nd, at 7.30 p.m. Particulars may be obtained from the Hon. Secretary, 106, Marlborough Lane, Charlton, London, S.E. 7.

World's Champion Cow.—A Holstein pure-bred "Carnation Ormsby Butter King," nine years old, in a 365 days' test, produced 38,606.6 lb. of milk and 1,752.5 lb. of butter. This record is more than eight times that of the average cow. During the year she consumed 11,750 lb. of fresh green feed, 4,380 lb. of beet pulp, 9,125 lb. sliced beets and 9,600 lb. silage.

Meat Consumption per capita in U.S.A.

	1935	1934
Beef and Veal	41.36 lb.	44.05 lb.
Pork	36.52	52.25
Lamb and Mutton	5.49	4.93
Lard	4.92	7.25

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